

be clinically acceptable for treatment while achieving critical serial organ dose constraints, with representative results shown in Figure 1. IMPT revealed significantly better PTV conformity ( $p=0.008$ ) and significant reductions in mean dose to several distant structures including larynx ( $p=0.002$ ) and brain ( $p=0.006$ ) against both VMAT and TomoHD plans.

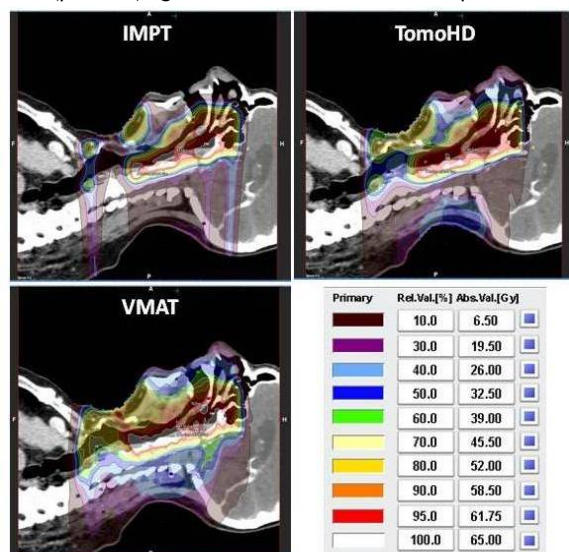


Fig. 1: Comparison of an intensity modulated proton therapy (IMPT), TomoTherapy (TomoHD) and a volumetric modulated arc therapy (VMAT) plans.

IMPT plans were more susceptible to anatomical changes compared to VMAT in nasal cavity filling simulation (e.g. mean reduction in CTV65 D98%: 1.9 vs 0.4 Gy) and weight loss (e.g. mean increase in cord D2%: 2.2 vs 0.2 Gy).

**Conclusions:** For the NPC cases studied, high quality plans were generated with all three techniques. IMPT demonstrated better PTV conformity and reduced dose to peripheral structures, the clinical significance of which is yet to be determined. On the other hand, IMPT was more susceptible to clinically observable anatomical changes.

---

Symposium with Proffered Papers: Around organs / combination therapy: Heart

---

#### SP-0120

##### Interaction of heart and lung after thoracic irradiation

P. Van Luijk<sup>1</sup>

<sup>1</sup>University Medical Center Groningen University of Groningen, Department Radiation Oncology, Groningen, The Netherlands

The aim of radiation oncology is to eradicate tumor tissue while attempting to preserve function of adjacent organs and tissues. In the treatment of thoracic tumors the heart is often at risk. Finding an optimum between the probability of curing the patient and the risk of inducing complications requires e.g. predictive models for the risk of such complications.

Current predictive models are generally based on one or more dose metrics, such as the (generalized) mean dose, volume receiving at least a defined minimum dose or the minimum dose to a defined fraction of the cardiac volume. The selection of these metrics, however, is generally based on assumed mechanisms and/or epidemiological parameter selection methods. As such, we hypothesized that a better understanding of mechanisms underlying cardiac damage may lead to improved selection of dose metrics for predictive models.

We performed a number of studies on mechanisms of cardiac toxicity using the irradiated rat as a model. In short, thoracic sub-volumes were irradiated using high-precision proton irradiation to yield varying irradiated cardiac and lung volumes. We analysed cardiac and pulmonary histological and function changes and found that heart irradiation already resulted in cardiac damage at 8 weeks post-irradiation [1]. Moreover, we found that this damage was aggravated if also the lung was irradiated [1,2]. Finally, we found that Captopril could both ameliorate the cardiac damage and its impact on lung damage and function. Therefore we concluded that the radiation response of heart and lung are closely related and cannot be regarded independently. As such, adding dosimetric and/or clinical parameters related to the lung in the development of predictive models for radiotherapy-induced cardiac damage may improve the accuracy of such model.

#### SP-0121

##### Experimental models of radiation-induced heart disease

M. Boerma<sup>1</sup>

<sup>1</sup>University of Arkansas for Medical Sciences, Pharmaceutical Sciences, Little Rock, USA

This presentation gives an overview of research performed in rat models of localized heart irradiation to address mechanisms by which ionizing radiation injures the heart. Rat models with mutations in the c-kit receptor gene have been used to investigate the role of mast cells in radiation-induced heart disease. The role of transforming growth factor-beta has been assessed by its pharmacological induction and the inhibition of its receptors. Moreover, rat models have been used to investigate whether the effects of radiation in the heart are different when radiation is combined with tyrosine kinase inhibitors, which have recently been associated with their own adverse cardiac effects.

#### SP-0122

##### Heart disease after radiotherapy

S.C. Darby<sup>1</sup>

<sup>1</sup>University of Oxford, Nuffield Department of Population Health, Oxford, United Kingdom

A number of epidemiological studies have recently provided insight into the magnitude of the risk of heart disease occurring as a result of radiotherapy and into the factors that affect the risk. In this talk, some of these results will be described.

#### OC-0123

##### Troponin T-release associates with cardiac radiation doses during adjuvant left-sided breast cancer radiotherapy

T. Skyttä<sup>1</sup>, S. Tuohinen<sup>2</sup>, E. Boman<sup>1</sup>, V. Virtanen<sup>2</sup>, P. Raatikainen<sup>3</sup>, P. Kellokumpu-Lehtinen<sup>1</sup>

<sup>1</sup>Tampere University Hospital, Oncology, Tampere, Finland

<sup>2</sup>Heart Center Co., Cardiology, Tampere, Finland

<sup>3</sup>Jyväskylä Central Hospital, Cardiology, Jyväskylä, Finland

**Purpose/Objective:** Adjuvant radiotherapy (RT) for left-sided breast cancer increases long term cardiac morbidity and mortality. No safe radiation threshold for heart is known. Troponin T is a sensitive marker of acute myocardial damage. Our aim was to evaluate the effects of left-sided breast cancer RT on high sensitivity cardiac troponin T (hs-cTnT) levels and possible associations with cardiac radiation doses and echocardiographic parameters.

**Materials and Methods:** A total of 58 patients with an early stage left-sided breast cancer or ductal carcinoma in situ